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Efficacy of intramuscular treatment of beef cows with oxytetracycline to reduce mastitis and to increase calf growth[1,2]

C. A. Lents*, R. P. Wettemann*[3], M. J. Paape†, J. A. Vizcarra*[4], M. L. Looper*[5], D. S. Buchanan*, and K. S. Lusby[6]

*Department of Animal Science, Oklahoma Agricultural Experiment Station, Stillwater 74078 and †Immunology and Disease Resistance Laboratory, USDA-ARS, Beltsville, MD 20705

ABSTRACT: Spring-calving multiparous Angus × Hereford cows were used to determine the efficacy of intramuscular treatment with oxytetracycline to reduce the incidence of mastitis-causing bacteria, decrease milk somatic cell counts (SCC), and increase calf growth. During 2 yr, milk samples were collected from each quarter from a total of 319 cows at 8 to 14 d after calving and at weaning, to determine the presence of bacteria and SCC. A California mastitis test (CMT) was performed on milk from each quarter of each cow at the initial sample collection. Cows with a CMT score of 1, 2, or 3 in at least one quarter, were randomly assigned to receive either an intramuscular injection of oxytetracycline (n = 63) or the control vehicle (n = 60), and cows with a CMT score of 0 or trace in all four quarters were not treated (n = 196). Calf weights were determined at birth, early lactation, and weaning. The number of somatic cells in milk and the percentage of quarters that were infected increased as CMT score increased ($P < 0.01$). The presence of mastitis-causing bacteria at calving increased ($P < 0.05$) the incidence of infection at weaning. The presence of mastitis-causing bacteria at weaning was associated with increased SCC for quarters and average SCC for cows ($P < 0.01$). Average SCC per cow at weaning increased ($P < 0.05$) as the number of infected quarters per cow increased. Treatment did not alter ($P > 0.10$) the percentage of cows or quarters infected with mastitis-causing bacteria or SCC of cows or quarters at weaning. Average SCC per cow was negatively correlated ($P < 0.05$) with calf weights at early lactation, but not with weaning weights of calves. Treatment did not influence ($P > 0.10$) calf weights at early lactation or at weaning. Cows with one or more dry quarters after calving had calves that weighed less at early lactation and weaning than cows with four functional quarters ($P < 0.01$). Intramuscular oxytetracycline treatment of beef cows that had CMT scores of 1 or greater after calving did not reduce intramammary infection rates or increase calf weights at weaning.

Key Words: Beef Cattle, Mastitis, Oxytetracycline, Somatic Cell Count, Weaning Weight

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Introduction

Milk production is the most important factor that influences weaning weights of calves (Neville, 1962; Rutledge et al., 1971). Mastitis decreases milk production in both dairy (Crossman et al., 1950; Bartlett et al., 1991; Lescourret and Coulon, 1994) and beef cows (Simpson et al., 1995). Intramammary infections in beef cows resulted in decreased weight gain of calves (Haggard et al., 1983; Watts et al., 1986; Newman et al., 1991) presumably due to decreased milk production. Treatment of beef cows for mastitis may increase weight gain of calves (Kirkbride, 1977; Newman et al., 1991).

Intramammary treatment of beef cows with an antibiotic at drying-off decreased the incidence of udder infections after the subsequent calving (Newman et al., 1991) and increased weight of calves at 60 d of age (Kirkbride, 1977). However, intramammary treatment of beef cows may not be feasible. Intramuscular treatment of dairy cows with systemic drugs, including oxytetracycline, maintained minimal inhibitory concentrations in the udder (Giesecke, 1977) and eliminated udder infections during lactation (Ziv and Storper, 1985) and the dry period (Soback et al., 1990). The objective of this study

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[3]Correspondence: 114 Anim. Sci. Bldg., Oklahoma State Univ. (phone: 405-744-6077; fax: 405-744-7390; E-mail: rpw@okstate.edu).

[4]Present address: Dept. of Animal Science and Food Technology, Texas Tech Univ., Lubbock, TX 79409.

[5]Present address: Animal Resources Dept., New Mexico State Univ., Las Cruces, NM, 88003-8003.

[6]Present address: Dept. of Animal Science, University of Arkansas, Fayetteville, AR, 72701.

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was to determine whether intramuscular oxytetracycline treatment of beef cows would decrease intramammary infection and increase calf growth.

Materials and Methods

Animals

Spring-calving multiparous Angus × Hereford cows (BW = 489 ± 3 kg; age = 6.0 ± 0.2 yr), with increased somatic cell counts (**SCC**) after calving, were used to determine the efficacy of intramuscular oxytetracycline to reduce the incidence of mastitis-causing bacteria, decrease milk SCC, and increase calf growth. Three hundred and nineteen cows were sampled in 2 yr (yr 1, n = 160; yr 2, n = 159) to identify cows with increased SCC after calving. Cows grazed bermuda grass pastures and native tallgrass range at the Oklahoma Agricultural Experiment Station Range Cow Research Center in north-central Oklahoma. During the winter, cows were group-fed 1.36 kg per animal of a 40% CP supplement daily to maintain a body condition score (**BCS**) of 4 to 5.5 (1 = emaciated and 9 = obese; Wagner et al., 1988). Cow BW was determined at 8 to 14 d after calving and at weaning. Weights of calves were determined at birth (yr 1, March 22 ± 2 d; yr 2, March 19 ± 2 d), early lactation (51 ± 6 d), and at weaning. Twenty control and 19 treated cows had their calves weaned early (69 ± 2 d of age). The remainder of the cows had calves weaned at 185 ± 9 d.

Milk Samples

Milk samples were collected from each quarter of each cow at 8 to 14 d after calving (postpartum sample) and at weaning. Calves were removed from cows for approximately 2 h before sampling. Cows were restrained in a squeeze chute and administered 10 I.U. of oxytocin (i.m.; Vedco, Inc., St. Joseph, MO) to facilitate milk let-down. Teats were dipped in a 0.1% iodine solution (Alfa Laval, Agri Inc., Kansas City, MO) and wiped dry with individual paper towels. The first two or three streams of milk were discarded and 10 mL of milk from each quarter were collected into plastic vials containing preservative (D & F Control Systems, Inc., San Ramon, CA). Teat ends were then individually disinfected with a cotton swab soaked in 70% ethyl alcohol. Two streams of milk from each quarter were discarded and 3 mL of milk was aseptically collected into sterile polypropylene snap cap tubes (Fisherbrand, Pittsburgh, PA). Samples (10 mL) were sent to the DHIA laboratory, Manhattan, KS, within 24 h for analyses of SCC. Somatic cell counts were quantified by a fluoro-opto-electronic method. Sterile samples were placed on ice and transported to the lab, stored at –20°C, and packaged in dry ice and transported to the Immunology and Disease Resistance Laboratory, USDA-ARS, Beltsville, MD, for bacteriological analyses.

Bacteriological Analyses

Bacteriological analyses were performed following standard procedures (NMC, 1990). Frozen, sterile milk samples were allowed to thaw at room temperature and vortexed. Twenty microliters of milk was plated on one quarter of a 100- × 15-mm Petri dish containing esculin blood agar (5% red blood cells), and on 100- × 15-mm Petri dishes containing mannitol and P-agar supplemented with acriflavine (Sigma Chemical Co., St. Louis, MO). Plates were incubated at 37°C, and bacterial growth was determined at 24 and 48 h.

A quarter of the udder was considered to be infected if three or more colonies of the same organism were isolated from the esculin blood agar plate. Identification of organisms was based on colony morphology, hemolytic and hydrolytic patterns, Gram stain (Bacto Gram Stain Set, Difco Laboratories, Detroit, MI), catalase production (hydrogen peroxide, Sigma Chemical Co.), and tube coagulase test (coagulase plasma EDTA, Difco Laboratories, Detroit, MI). Tube coagulase tests were incubated at 37°C and examined for clot formation at 4 and 24 h. *Staphylococcus aureus* was identified by selective growth on P-agar supplemented with acriflavine (Sigma Chemical Co.) and fermentation of mannitol. Coagulase-negative staphylococci were further identified using an identification system for staphylococci (Api Staph, API-Bio Mérieux Vitex, Inc., Hazelwood, MO). Bacteria were passed four times on esculin blood agar plates, and a second coagulase test was performed before Api Staph wells were inoculated. Streptococcal isolates were identified using CAMP-esculin plates (Wilson et al., 1971), made in accordance with standard procedures (NMC, 1987). *Corynebacterium bovis* and *Bacillus* spp. were identified by time of appearance on incubated plates, colony morphology, and Gram stain.

California Mastitis Test

After collection of milk component and bacteriological samples, a California mastitis test (**CMT**) was performed for each quarter of each cow. A stream of milk from each quarter was discarded, and approximately 5 mL of milk was collected from each quarter into a corresponding cup on the CMT paddle (Dairy Research Products Inc., Spencerville, IN). After sampling, teats were dipped in 0.1% iodine solution. The paddle was tilted to allow excess milk to drain from each cup without mixing with other samples, and approximately 2 mL of milk remained in each receptacle. An equal volume of CMT reagent (Dairy Research Products, Inc.) was added to each cup and the paddle was gently swirled to mix the samples. A CMT score of 0, trace, 1, 2, or 3, as described by Schalm and Noorlander (1957), was assigned to each sample within 2 min of collection, based on the amount of precipitant and gel formation. Cows with a CMT score of 0 or trace in all four quarters were not treated (n = 196). Cows with a CMT score of 1, 2, or 3 in at least one quarter were blocked by CMT score and calving date,

and randomly received either an intramuscular injection of oxytetracycline (n = 63) or control vehicle (n = 60) within 4 min after the CMT sample was taken.

Treatment

Each milliliter of antibiotic contained 200 mg of oxytetracycline (Liquamycin LA200, Animal Health Division of Pfizer, Inc., NY), and on a weight-to-volume basis, 40% 2-pyrrolidone and 5% polyvinlypyrrolidone in a sterile aqueous solution. This is a formulation that delivers 3 d of sustained antibiotic therapy. The control vehicle consisted of 40% 2-pyrrolidone (BASF Corp., Parisppany, NJ.) and 5% polyvinlypyrrolidone (Aldrich Chemical Co., Inc., Milwaukee, WI) in a sterile aqueous solution. Cows received 1 mL of the assigned treatment per 10 kg of BW. Intramuscular injections were administered along the lateral surfaces of the dorsal one-third of the neck. Only 10 mL of solution was administered per injection site, with no more than three injection sites per side of the neck.

Statistical Analyses

Somatic cell counts ($\times 10^3$ cells/mL) were analyzed using log-transformed values (Ali and Shook, 1980); however, geometric means for actual somatic cell values are reported (Shook, 1982). Log-transformed SCC greater or less than three standard deviations from the mean (n = 2) were considered outliers and deleted. Average SCC of the four quarters for each cow were determined as a geometric mean and used as the mean SCC value for each cow. Least squares analyses of variance were used to determine treatment effect on SCC and infection status at weaning. The model included treatment, postpartum infection status, year, and significant ($P < 0.10$) interactions. A cow was classified as infected if one or more mastitis-causing organism was isolated in any quarter. Chi-square was used to determine the effects of treatment and infection status at calving on the percentage of cows and quarters infected at 69 d and weaning (185 d). Separate statistical analyses were performed for cows that had calves weaned at early lactation.

Regression analyses were used to determine the relationships between SCC (independent variable) and CMT score (dependent variable). Due to a significant year effect, analyses were performed within year. For statistical analysis, CMT scores of 0, trace, 1, 2, and 3 were assigned values of 0, 1, 2, 3, and 4 respectively. Indicator (dummy) regression analyses were used to determine whether slopes and intercepts for regression lines for each year were different (Neter et al., 1989; Steel et al., 1997).

Only calves that were weighed at 51 d were included in the analysis of early lactation weight. Data from calves that were greater or less than one standard deviation from the mean age were excluded so that the analysis would consist of calves of a similar age. Weights for 66 calves nursing cows with a CMT ≥ 1 were analyzed using least squares analysis of variance to determine effects of treatment and postpartum infection status on calf weights at early lactation. The model included treatment, postpartum infection status, calf sex, and year, with cow age, calf age, and birth weight as covariates. The relationships of CMT status (< 1 or ≥ 1) at the postpartum sample and treatment with birth weight of 319 calves were determined with least squares analysis of variance. The model included CMT status and treatment, sex of calf, and year, with cow age as a covariate. Weights for 169 calves were analyzed with least squares analysis of variance to determine the effect of CMT status at the postpartum sample and treatment on calf weight at early lactation. The model included CMT status and treatment, sex of calf, and year, with cow age, calf age, and birth weight as covariates. The relationship between calf weight and average SCC postpartum was determined by partial correlation adjusted for year. Analyses of early lactation weights (51 d) included calves weaned early and those weaned at the end of lactation.

The effects of postpartum infection status and treatment on weaning weight of calves adjusted to 205 d were determined using a model that included year, postpartum infection status, treatment, calf sex, and all interactions, with cow age as a covariate. The effects of CMT status at the postpartum sample and treatment on weaning weight of calves (n = 216) adjusted to 205 d were determined using a model that included CMT status and treatment, year, calf sex, and the interactions, with cow age as a covariate. Relationship between average SCC and weaning weights was determined by partial correlations adjusted for year. Analysis of weaning weights included only calves weaned at approximately 185 d of age.

The effect of dry quarters after calving on calf growth at 51 d and weaning was assessed. For calf weights at 51 d, the model included year, calf sex, dry quarters postpartum, and the interaction, with calf age, birth weight, and cow age as covariables. The model for 205-d adjusted weaning weights included year, calf sex, dry quarters postpartum, and the interactions, with cow age as a covariate.

Results

The mastitis-causing bacteria isolated in this experiment were *Staphylococcus aureus, Corynebacterium bovis,* and coagulase-negative staphylococci. Coagulase-negative staphylococci included *Staphylococcus hyicus, Staphylococcus chromogenes, Staphylococcus epidermidis, Staphylococcus cohnii, Staphylococcus warneri,* and *Staphylococcus hominis.* The percentages of cows and quarters that were infected at calving were 37.3 and 15.2%, respectively. Of those, 13.8% of cows and 5.2% of quarters were infected with *Staphylococcus aureus;* 25.5% of cows and 8.6% of quarters were infected with coagulase-negative staphylococci; 3.1% of cows and 1.4% of quarters were infected with *Corynebacterium bovis.* Five percent of cows were infected with more than one

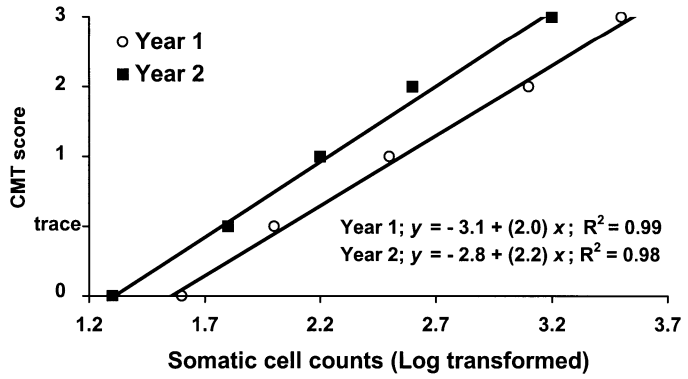

Figure 1. Least squares means (symbols) and least squares regressions (lines) for quarter somatic cell counts with California mastitis test (CMT) scores in beef cows after calving. At 8 to 14 d postpartum, a CMT was performed for each quarter of each cow. For statistical analysis, CMT scores of 0, trace, 1, 2, and 3 were assigned values of 0, 1, 2, 3, and 4, respectively.

organism, but multiple species of bacteria were not found in any quarter.

The intercepts for the regressions of CMT on SCC for yr 1 and 2 were different ($P < 0.01$), but the slopes were similar. In both years, CMT score increased with increasing SCC ($P < 0.001$; Figure 1). A total of 981, 80, 82, 55, and 47 quarters had CMT scores of 0, trace, 1, 2, and 3, respectively. The percentage of quarters infected increased with increasing CMT score (Table 1). The percentage of infected quarters was not different for quarters with CMT scores of 0 or trace ($P > 0.10$). More quarters with a CMT score of 1 were infected than quarters that had a CMT score of 0 ($P < 0.05$). The percentages of quarters with CMT scores of 2 or 3 that were infected were greater than for any other score ($P < 0.01$). Quarter SCC after calving in yr 1 were greater than in yr 2 ($P < 0.0001$), but there was not a CMT score × year effect on SCC.

Treatment and year did not influence the percentage of cows or quarters infected at weaning ($P = 0.50$; Table 2), and there was no interaction between treatment or year and infection status at the postpartum sample. Pooled across years, 39% of cows and 18% of quarters that were treated postpartum were infected at weaning, compared with 38% of control cows and 14% of control quarters. More cows infected with mastitis-causing bacteria at 8 to 14 d after calving were infected at weaning and had more infected quarters at weaning than cows that were uninfected at the postpartum sample (Table 3). Fifty-one percent of cows that were infected postpartum were infected at weaning, and 28% of cows not infected postpartum were infected at weaning ($P < 0.05$). Similarly, 35% of quarters that were infected postpartum

Table 1. Percentage of quarters that were infected and somatic cell counts (SCC) of quarters that were classified as 0, trace, 1, 2, or 3 by the California mastitis test (CMT)

	CMT Score[a]					
Item	0	trace	1	2	3	SEM
Infected quarters, %	11[b]	16[bc]	25[c]	40[d]	44[d]	—
SCC, × 10^3 cells/mL[a]	66[b]	194[c]	510[d]	1,408[e]	3,035[f]	61

[a]CMT score: 0 = no precipitate, trace = slight precipitate, 1 = distinct precipitate, 2 = gel forms, 3 = complete coagulation. A CMT was performed on milk from each quarter of each cow 8 to 14 d after calving.
[b,c,d,e,f]Means within a row with different superscripts differ ($P < 0.05$).

Table 2. Effects of treatment on infection and somatic cell counts (SCC) of cows and quarters at weaning

	Treatment[ab]		
Item	Control	Treated	SEM
No.			
Cows	40	44	—
Quarters	154	166	—
Infected at weaning[c]			
Cows, %	38	39	—
Quarters, %	14	18	—
SCC at weaning, × 10^3 cells/mL			
Cow average[d]	247	311	60
Quarter	501	524	86

[a]Cows were administered control solution or oxytetracycline at 8 to 14 d after calving.
[b]Means within a row do not differ ($P > 0.10$).
[c]Cows and quarters were defined as infected if they had the presence of one or more mastitis-causing organisms.
[d]Geometric mean for somatic cell counts of all quarters of a cow.

Table 3. Influence of infection status of cow or quarter at the time of treatment on the percentage of cows and quarters infected at weaning, and weight of calves at early lactation and weaning

	Infection status at treatment[ab]		
Item	Uninfected	Infected	SEM
No.			
Cows	47	37	—
Quarters	255	53	—
Percentage infected at weaning			
Cows	28[d]	51[e]	—
Quarters	13[f]	35[g]	—
Weight of calves, kg			
Early lactation (51 d)	81	81	2
Weaning (205 d adjusted)[c]	231	223	5

[a]Cows were administered control solution or oxytetracycline at 8 to 14 d after calving.
[b]Cows and quarters were defined as infected if they had the presence of one or more mastitis-causing organisms.
[c]Weight of calves at weaning (185 d) was adjusted to 205 d.
[d,e]Means within a row with different superscript letters differ ($P < 0.05$).
[f,g]Means within a row with different superscript letters differ ($P < 0.01$).

Table 4. Effect of infection status of cow or quarter on somatic cell counts at weaning

Item	Infection status at weaning[ab] Uninfected	Infected	SEM
Somatic cell counts, × 10^3 cell/mL			
Cow			
Average SCC[c]	54[e] (52)	288[f] (32)	73
Quarter[d]			
Year 1	75[e] (150)	1571[f] (34)	99
Year 2	106[e] (107)	583[f] (20)	125

[a]Number of observations in parentheses.
[b]Cows and quarters were defined as infected if they had the presence of one or more mastitis-causing organisms.
[c]Average SCC = geometric mean for somatic cell counts of all quarters of a cow.
[d]Year × infection status ($P < 0.01$).
[e,f]Means within a row with different superscript letters differ ($P < 0.05$).

Table 5. Effect of number of dry quarters after calving on weight of calves at early lactation and weaning

Calf weight, kg	Number of dry quarters after calving 0	≥1	SEM
Early lactation, 51 d[a]	83[c] (59)	67[d] (7)	3
Weaning, 205 d adjusted[ab]	233[c] (71)	202[d] (13)	6

[a]Numbers of observations (cows) in parentheses.
[b]Weight of calves at weaning (185 d) was adjusted to 205 d.
[c,d]Means within a row with different superscript letters differ ($P < 0.01$).

were infected at weaning, and 13% of quarters that were not infected postpartum were infected at weaning ($P < 0.01$).

There was not a treatment × postpartum infection status effect on average SCC ($P = 0.20$). Average SCC at weaning was not different ($P = 0.40$) for control and treated cows (Table 2). Somatic cell count per quarter at weaning was not different for control and treated quarters ($P = 0.30$; Table 2). The presence of mastitis-causing bacteria at weaning was associated with increased average SCC at weaning ($P < 0.05$; Table 4). Infected cows had greater average SCC than noninfected cows ($288 \pm 82 \times 10^3$ vs $54 \pm 63 \times 10^3$ cells/mL, respectively).

There was a year × infection status effect on SCC per quarter at weaning ($P < 0.01$; Table 4). Somatic cell counts for infected and uninfected quarters were not different between years. Infected quarters had greater SCC than uninfected quarters in both years, but in yr 1, the increase in SCC with infection was greater ($P < 0.01$) than in yr 2. Cows with more infected quarters at weaning had greater average SCC at weaning ($P < 0.01$). Cows with no infection, or only one infected quarter had similar average SCC ($54 \pm 63 \times 10^3$ and $65 \pm 116 \times 10^3$ cells/mL, respectively). However, average SCC of cows with two or more infected quarters were greater ($484 \pm 107 \times 10^3$ cells/mL; $P < 0.01$) than for cows with 0 or 1 infected quarters.

Postpartum infection status of cows tended ($P = 0.09$) to alter infection status at 69 d. Forty-one percent of cows infected after calving were infected at 69 d compared with 8% of cows that were not infected after calving. Postpartum infection status of quarters affected infection status at 69 d ($P < 0.01$). Twenty-two percent of quarters that were infected postpartum were infected at 69 d compared with 7% of uninfected quarters that were infected at early lactation. The percentage of cows and quarters infected at 69 d was similar for treated and controls ($P = 0.50$). Treatment did not influence average SCC or SCC per quarter at 69 d of lactation ($P = 0.30$).

Cows with one or more dry quarters after calving had calves that weighed 16 kg less at 51 d than cows with no dry quarters ($P < 0.01$; Table 5). Cows that were not infected at 8 to 14 d after calving had calves with similar weights ($P = 0.90$) at 51 d postpartum as cows infected with mastitis-causing organisms (Table 3). Calves in yr 1 weighed less at 51 d of lactation than calves in yr 2. There were no treatment × infection status or year interactions ($P = 0.70$) for calf weights at 51 d of lactation. Calf weights were similar ($P = 0.40$) at 51 d of lactation among control and treated cows that had CMT scores of 1 or greater postpartum, and cows that had CMT scores less than 1 (Table 6). Average SCC of all cows at the postpartum sample was negatively correlated with weights of calves at early lactation (adjusted for year; $r = -0.26$; $P < 0.05$).

Calves weighed more at weaning in yr 2 than in yr 1 (240 ± 5 vs 215 ± 5 kg respectively; $P < 0.01$), but there were no interactions of main effects with year. Weaning weights were similar ($P = 0.20$) among control and treated cows that had CMT scores of 1 or greater postpartum and cows that had CMT scores less than 1 (Table 6). The presence of mastitis-causing organisms in the udder of cows at calving did not influence weaning weights ($P = 0.30$; Table 3). Cows with one or more dry quarters had calves that weighed 31 kg less ($P < 0.01$) at weaning compared with calves from cows with no dry quarters (Table 5).

Discussion

Somatic cell counts of dairy cows are highly correlated with CMT scores (Pearson and Greer, 1974). We observed a similar relationship between CMT scores and SCC in beef cows; however, the values for SCC of beef cows (range of 66 to $3{,}035 \times 10^3$ cells/mL) are less than those for dairy cows (Schalm and Noorlander, 1957; Philpot and Nickerson, 1991). The use of the CMT for beef production has been limited. Factors that influenced CMT scores in beef herds have been reported (Wilson et al., 1971); however, SCC associated with CMT scores were not included. Newman et al. (1991) used CMT to screen quarters of beef cows for mastitis treatment, but

Table 6. Effect of increased CMT postpartum and treatment on weight of calves at early lactation and weaning

	Treatment[a] of cows with increased CMT			
Calf weight, kg	Control	Treated	Normal CMT	SEM
Birth	39 (60)[bc]	36 (63)[c]	41 (196)[d]	1
Early lactation, 51 d[c]	84 (34)	80 (32)	84 (103)	2
Weaning, 205 d adjusted[d]	231 (40)	223 (44)	229 (132)	4

[a]Cows were administered control solution or oxytetracycline at 8 to 14 d after calving.
[b]Numbers of observations in parentheses.
[cd]Means within a row with different superscript letters differ ($P < 0.01$).

SCC were not reported. To our knowledge, this study is the first to describe the relationship between SCC and CMT scores of beef cows.

The percentage of quarters infected with any organism postpartum averaged 17% for CMT scores 0 through 1, which was less than the 42% found for CMT scores 2 and 3. In dairy cows, only CMT scores of 2 and 3 are considered reliable to predict infection, with trace and 1 classified as suspect (Gray and Schalm, 1960). Percentage of infection in dairy cows ranges from 33 to 54% for quarters with a CMT score of 1, and up to 71 to 96% for quarters with a CMT score of 3 (Marshall and Edmondson, 1962; Pearson and Greer, 1974). In beef cows, 8, 21, 45, 60, and 71% of quarters with CMT scores of 0, trace, 1, 2, and 3 were infected (Newman et al., 1991), which is greater than the percentage of infected quarters in our experiment with similar CMT scores. In our experiment, CMT scores of 2 and 3 for quarters without infection probably resulted from stage of lactation that samples were taken. Somatic cell counts are usually greatest during early and late lactation in dairy (Bodoh et al., 1976; Reneau, 1986) and beef cows (Wilson et al., 1971; Hunter and Jeffrey, 1975; Newman et al., 1991), and SCC may be elevated for up to 2 wk after parturition (Cullen, 1968; Natzke et al., 1972). We sampled at 8 to 14 d postpartum to evaluate CMT, whereas Newman et al. (1991) took samples for CMT at 4 wk postpartum. In addition, the relationship between CMT and infection status in this experiment was based on only a single milk sample. In the current study, CMT scores were correlated with SCC, but not infection.

The presence of mastitis-causing bacteria after calving was associated with an increased incidence of infection at weaning. Fourteen to 31% of quarters of dairy cows remained infected throughout lactation (Jackson, 1961). Intramammary infections in beef cows tend to persist throughout lactation (Newman et al., 1991), and Simpson et al. (1995) determined that 39% of infected quarters of beef cows remained infected.

Intracellular location of some mastitis causing organisms can result in decreased effectiveness of intramammary treatment (Nickerson and Owens, 1993). Thus, systemic treatment of mastitis has been evaluated (Giesecke, 1977; Ziv, 1980). Intramuscular treatment is as effective as intramammary treatment in eliminating mastitis in dairy cows (Ziv and Storper, 1985; Jarp et al., 1989). A combination of intramuscular and intramammary treatment increased cure rates and maintained greater concentrations of antibiotics in the mammary tissue (Owens et al., 1988; 1994; Erskine et al., 1994). Oxytetracycline can maintain minimal inhibitory concentrations in udder secretions and provide protection against new infection (Giesecke, 1977; Soback et al., 1990). In a previous study with beef cows, we determined that intramuscular oxytetracycline treatment of all cows after calving did not decrease udder infection at weaning (Duenas et al., 2001). However, no attempt was made to identify infected cows before treatment, thus the inclusion of uninfected cows could have negated any possible benefit of treatment. In the current study, cows were selected for treatment on the basis of CMT score, however treatment did not decrease the percentage of infected cows. Reasons for the lack of efficacy of oxytetracycline are not readily apparent. In dairy cows, duration of treatment has the greatest impact on cure rate (Jarp et al., 1989). A single injection after calving maybe too short a duration of treatment to decrease bacterial populations. Another possibility is that treatment may cure infections over a short period, but cows may become reinfected prior to the subsequent sampling period. However, no treatment effects were found on infection rates of cows at early lactation. Furthermore, antibiotic sensitivity of isolated bacteria was not performed, and some resistant strains of bacteria may have been present. We defined a cow to be infected based on the presence of mastitis causing bacteria. No attempt was made to quantify bacteria for each quarter, and not all cows that were defined to be infected had clinical mastitis. Treatment may reduce bacterial populations and improve udder health, but not completely eradicate the organisms.

Intramammary infection is the major factor that contributes to an increase in SCC in dairy cows (Dohoo and Meek, 1982; Reneau, 1986; Harmon, 1994). Both experimentally induced and naturally occurring infections result in increased SCC (Sheldrake et al., 1983; Fox and Schultz, 1985; Schukken et al., 1994). Our results with beef cows revealed that average SCC per cow was greater for infected cows compared with uninfected cows. This is in agreement with other reports for beef cows (Newman et al., 1991; Simpson et al., 1995; Paape et al., 2000). Hunter and Jeffrey (1975) determined that most infected quarters of beef cows had SCC greater

than 500×10^3 cells/mL, and 20% had SCC greater than $1,000 \times 10^3$ cells/mL. While individual quarters that were infected had abnormal SCC, cows usually had SCC of less than 500×10^3 cells/mL (Brown et al., 1998; Duenas et al., 2001). This is in contrast to other investigators who found that SCC of uninfected quarters were 555×10^3 cells/mL while infected quarters had SCC greater than 794×10^3 cells/mL (Watts et al., 1986; Simpson et al., 2000). In our study, uninfected quarters had SCC of 91×10^3 cells/mL while infected quarters had SCC of $1,077 \times 10^3$ cells/mL. This agrees with Newman et al. (1991) who found that uninfected quarters of beef cows had SCC of about 20×10^3 cells/mL. Wilson et al. (1971) observed that although mastitis causing bacteria may be present in beef cows, SCC usually were not elevated to an abnormal concentration.

Milk loss of dairy cows is 7 to 9% when SCC are greater than 400×10^3 cells/mL (Schultz, 1977; Miles et al., 1992). Average SCC per cow were negatively correlated with weight of calves at early lactation, but not weaning weight of calves. Decreased weights at early lactation were presumably due to decreased milk production. Primiparous Simmental cows with increased SCC produced less milk than cows with fewer SCC (Simpson et al., 1995). Others found that infection with major mastitis causing bacteria increased SCC which was associated with decreased calf weights (Watts et al., 1986; Newman et al., 1991).

Intramammary infections of beef cows decreased weaning weights of calves by 7 to 9% (Haggard et al., 1983; Watts et al., 1986). In other studies, growth of calves was influenced the most by the infection status of dams at 60 to 100 d after calving (Newman et al., 1991). We found that infection status of the cow at the postpartum sample did not alter weight of calves at 51 d of lactation or weaning. This lack of detrimental influence of udder infection on calf weight gain could be due to the fact that not all cows defined as infected had clinical mastitis, and milk production may not have been compromised severely enough to cause an effect on calf weights. In addition, udder infections may decrease milk production during early lactation without a major influence on calf weight gain. A calf may not consume all milk that is secreted early in lactation (Newman et al., 1991), thus reduced milk secretion of the dam early in the life of the calf may not adversely affect gain. As calves get older, they receive energy from sources other than milk (Neville, 1962; Haggard et al., 1983; Ansotegui et al., 1991). Therefore, the effect of decreased milk production due to udder infections may not be apparent over the entire lactation. Furthermore, most cows were infected in only one quarter, and this may not produce a large enough decrease in milk production to adversely effect calf growth. We previously determined that calf weights at early-lactation and weaning were decreased when calves nursed cows with three or more infected quarters (Duenas et al., 2001).

Newman et al. (1991) determined that treating beef cows with intramammary antibiotics decreased udder infections, but did not evaluate the effects on weight gain of calves. Others report that calves from beef cows treated for mastitis weighed 12.5% more at 60 d of age than calves from untreated controls (Kirkbride, 1977). Intramuscular treatment of cows with antibiotics at drying-off and(or) after calving did not alter weaning weights of calves (Duenas et al., 2001). In the current study, treating beef cows that had increased SCC after calving with antibiotics did not alter calf weights at early lactation or weaning. This agrees with the observation that treatment did not alter infection status of cows, and that infection status did not adversely influence weight gain of calves. With a highly variable trait such as weaning weight, a greater number of observations or the use of weaning weight EPD as a covariate may be required to detect a treatment effect.

In this experiment, 4.1% of cows had dry quarters at weaning. This is slightly less than the 10.2% of cows with dry quarters previously reported for this herd (Duenas et al., 2001). Calves that nursed cows with one or more dry quarters weighed less at 51 d and 205 d than calves nursing cows with four functional quarters. This is similar to our previous report that calves weighed 17 kg less at 60 d and 25 kg less at 205 d when they nursed dams that had one or more dry quarters (Duenas et al., 2001).

In conclusion, CMT is a better indicator of SCC than an indicator of infection. Average SCC in milk from cows on d 8 to 14 of lactation was negatively correlated with calf weight at 51 d of lactation, but not at weaning. Weaning weights of calves were reduced when cows had one or more dry quarters after calving. Intramammary infections of beef cows caused increased SCC, but did not adversely influence weight gain of calves. Intramuscular oxytetracycline treatment of beef cows, that had CMT scores of 1 or greater at 8 to 14 d after calving, did not alter SCC, infection rates, or calf weights at weaning.

Implications

Treatment of beef cows with intramuscular oxytetracycline after calving does not decrease somatic cell counts in milk, or decrease intramammary infection at weaning. Weight of calves at 51 d of lactation and weaning is not influenced by intramuscular oxytetracycline treatment of cows after calving, but weights are significantly reduced if cows have one or more dry quarters. The CMT is a reliable cow-side method to asses SCC in beef cows. Methods should be developed to decrease the incidence of dry quarters, and producers should use weaning weights of calves to help identify and cull cows that have nonfunctional quarters.

Literature Cited

Ali, A. K. A., and G. E. Shook. 1980. An optimum transformation for somatic cell concentration in milk. J. Dairy Sci. 63:487–490.

Ansotegui, R. P., K. M. Havstad, J. D. Wallace, and D. M. Hallford. 1991. Effects of milk intake on forage intake and performance of suckling range calves. J. Anim. Sci. 69:899–904.

Bartlett, P. C., J. V. Wijk, D. J. Wilson, C. D. Green, G. Y. Miller, G. A. Majewski and L. E. Heider. 1991. Temporal patterns of lost milk production following clinical mastitis in a large Michigan Holstein herd. J. Dairy Sci. 74:1561–1572.

Bodoh, G. W., W. J. Battista, L. H. Schultz, and R. P. Johnston. 1976. Variation in somatic cell counts in dairy herd improvement milk samples. J. Dairy Sci. 59:1119–1123.

Brown, M. A., I. Duenas, M. Paape, W. G. Jackson, J. R. Miesner, and A. H. Brown. 1998. Incidence of mastitis-causing organisms in Angus, Brahman, and reciprocal-cross cows grazing endophyte-infected tall fescue or common bermudagrass. Prof. Anim. Sci. 14:127–132.

Crossman, J. V., F. H. Dodd, J. M. Lee, and F. K. Neave. 1950. The effect of bacterial infection on the milk yield of the individual quarters of the cow's udder. J. Dairy Res. 17:128–158.

Cullen, G. A. 1968. Cell counts throughout lactation. Vet. Rec. 83:125–128.

Dohoo, L. R., and A. H. Meek. 1982. Somatic cell counts in bovine milk. Can. Vet. J. 23:119–125.

Duenas, M. I., M. J. Paape, R. P. Wettemann, and L. W. Douglass. 2001. Incidence of mastitis in beef cows after intramuscular administration of oxytetracycline. J. Anim. Sci. 79:1996–2005.

Erskine, R. J., P. C. Bartlett, P. C. Crawshaw, and D. M. Gombas. 1994. Efficacy of intramuscular oxytetracycline as a dry cow treatment for *Staphylococcus aureus* mastitis. J. Dairy Sci. 77:3347–3353.

Fox, L. K., and L. H. Schultz. 1985. Effect of infection status on quarter milk production and composition following omitted milking. J. Dairy Sci. 68:418–423.

Giesecke, W. H. 1977. The systemic therapy of clinical bovine mastitis. J. S. Afr. Vet. Assoc. 48:289–291.

Gray, D. M., and O. W. Schalm. 1960. California Mastitis Test Results. J. Am. Vet. Med. Assoc. 136:195–198.

Haggard, D. L., R. J. Farnsworth, and J. A. Springer. 1983. Subclinical mastitis of beef cows. J. Am. Vet. Med. Assoc. 182:604–606.

Harmon, R. J. 1994. Physiology of mastitis and factors affecting somatic cell counts. J. Dairy Sci. 77:2103–2112.

Hunter, A. C., and D. C. Jeffrey. 1975. Subclinical mastitis in suckler cows. Vet. Record. 96:442–447.

Jackson, R. A. 1961. A practical mastitis control program. Vet. Med. 56:143–146.

Jarp, J., H. P. Bugge, and S. Larsen. 1989. Clinical trial of three therapeutic regimens for bovine mastitis. Vet. Record 124:630–634.

Kirkbride, C. A. 1977. Mastitis in beef cows. J. Am. Vet. Med. Assoc. 170:1141–1142.

Lescourret, F., and J. B. Coulon. 1994. Modeling the impact of mastitis on milk production by dairy cows. J. Dairy Sci. 77:2289–2301.

Marshall, R. T., and J. E. Edmondson. 1962. Value of California Mastitis Test records to the practitioner. J. Am. Vet. Med. Ass. 140:45–49.

Miles, H., W. Lesser, and P. Sears. 1992. The economic implications of bioengineered mastitis control. J. Dairy Sci. 75:596–605.

National Mastitis Council. 1987. Laboratory and field handbook on bovine mastitis. Arlington, VA.

National Mastitis Council. 1990. Microbiological procedures for the diagnosis of bovine udder infection (3rd Ed.). Arlington, VA.

Natzke, R. P., R. W. Everett, and D. S. Postle. 1972. Normal milk somatic cell counts. J. Milk Food Technol. 35:261–263.

Neter, J., M. H. Kutner, C. J. Nachtsheim, and W. Wasserman. 1989. Applied linear regression models (3rd ed.). Richard D. Irwin, Inc. Chicago, Il., pp 455–496.

Neville, W. E. 1962. Influence of dam's milk production and other factors on 120- and 240- day weight of Hereford calves. J. Anim. Sci. 21:315–320.

Newman, M. A., L. L. Wilson, E. H. Cash, R. J. Eberhart, and T. R. Drake. 1991. Mastitis in beef cows and its effects on calf weight gain. J. Anim. Sci. 69:4259–4272.

Nickerson, S. C., and W. E. Owens. 1993. Staphylococcus aureus mastitis: Reasons for treatment failures and therapeutic approaches for control. Agri. Pract. 14:18–23.

Owens, W. E, J. L. Watts, R. L. Boddie, and S. C. Nickerson. 1988. Antibiotic treatment of mastitis: Comparison of intramammary and intramammary plus intramuscular therapies. J. Dairy Sci. 71:3143–3147.

Owens, W. E., S. C. Nickerson, J. L. Watts, R. A. Rzepkowski, and R. J. Yancey. 1994. Milk, serum, and mammary tissue concentration of Pirlimycin following intramuscular, intramammary, or combination therapy of chronic *Staphylococcus aureus* mastitis. Agri. Practice 15(3):19–23.

Paape, M. J., M. I. Duenas, R. P. Wettemann, and L. W. Douglass. 2000. Effects of intramammary infection and parity on calf weaning weight and milk quality in beef cows. J. Anim. Sci. 78:2508–2514.

Pearson, J. K. L., and D. O. Greer. 1974. Relationship between somatic cell counts and bacterial infections of the udder. Vet Rec. 95:252–257.

Philpot, W. N., and S. C. Nickerson. 1991. Mastitis: Counter attack. Babson Bros. Co., Naperville, IL.

Reneau, J. K. 1986. Effective use of dairy herd improvement somatic cell counts in mastitis control. J. Dairy Sci. 69:1708–1720.

Rutledge, J. J., O. W. Robison, W. T. Ahlschwede, and J. E. Legates. 1971. Milk yield and its influence on 205-day weight of beef calves. J. Anim. Sci. 33:563–567.

Schalm, O. W., and D. O. Noorlander. 1957. Experiments and observations leading to development of the California Mastitis Test. J. Am. Vet. Med. Assoc. 130:199–204.

Schukken, Y. H., B. A. Mallard, J. C. M. Dekkers, K. E. Leslie, and M. J. Stear. 1994. Genetic impact on the risk of intramammary infection following *Staphylococcus aureus* challenge. J. Dairy Sci. 77:639–647.

Schultz, L. H. 1977. Somatic cells in milk-physiological aspects and relationship to amount and composition of milk. J. Food Prot. 40:125–131.

Sheldrake, R. F., R. J. T. Hoare, and G. D. McGregor. 1983. Lactation stage, parity, and infection affecting somatic cells, electrical conductivity, and serum albumin in milk. J. Dairy Sci. 66:542–547.

Shook, G. E. 1982. Approaches to summarizing somatic cell count which improve interpretability. In: Proc. 21st Annu. Mtg. Natl. Mastitis Counc., Louisville, KY. p 150. Natl. Mastitis Counc. Inc., Arlington, VA.

Simpson, R. B., S. C. Nickerson, J. D. Shockey, and J. M. Phillips. 2000. Prevalence of mastitis pathogens preceding and following parturition in primiparous beef cows. Prof. Anim. Sci. 16:188–195.

Simpson, R. B., D. P. Wesen, K. L. Anderson, J. D. Armstrong, and R. W. Harvey. 1995. Subclinical mastitis and milk production in primiparous Simmental cows. J. Anim. Sci. 73:1552–1558.

Soback, S., G. Ziv, M. Winkler, and A. Saran. 1990. Systemic dry cow therapy—A preliminary report. J. Dairy Sci. 73:661–666.

Steel, R. G. D., J. H. Torrie, and D. A. Dickey. 1997. Principles and procedures of statistics: A biometrical approach (3rd ed.). McGraw-Hill Inc. New York.

Wagner, J. J., K. S. Lusby, J. W. Oltjen, J. Rakestraw, R. P. Wettemann, and L. E. Walters. 1988. Carcass composition in mature Hereford cows: estimation and effect on daily metabolizable energy requirement during winter. J. Anim. Sci. 66:603–612.

Watts, J. L., J. W. Pankey, W. M. Oliver, S. C. Nickerson, and A. W. Lazarus. 1986. Prevalence and effects of intramammary infection in beef cows. J. Anim. Sci. 62:16–20.

Wilson, L. L., R. J. Eberhart, M. J. Simpson, H. Varela-Alvarez, M. C. Rugh, and L. G. Bair. 1971. Incidence of intramammary infections and effects of number of lactations, lactation stage, quarter and calf sex on somatic cell content of milk from Angus-Holstein F_1 cows. J. Anim. Sci. 33:433–437.

Ziv, G. 1980. Drug selection and use in mastitis: Systemic vs local therapy. J. Am. Vet. Med. Assoc. 176:1109–1115.

Ziv, G., and M. Storper. 1985. Intramuscular treatment of subclinical staphylococcal mastitis in lactating cows with penicillin G, methicillin and their esters. J. Vet. Pharmacol. Therap. 8:276–283.